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**Some Points in the Diagnosis and Localization of Cerebral Abscess.**

By C. P. SYMONDS, M.D.

IN this paper I shall confine myself to cerebral abscess arising from suppuration in the ear, and situated entirely within the dural membrane. I shall only refer briefly to those symptoms and signs of abscess in the cerebellar and temporal lobes which are of chief value in making an early diagnosis and localization. I shall then describe in greater detail two less common conditions, the superficial abscess, and the localized non-suppurative encephalitis.

## CEREBRAL ABSCESS.

*Temporal and Cerebellar.*

In the early stages the symptoms are, as a rule, those common to retained pus elsewhere; fever, a rapid pulse, general malaise and anorexia. Headache may be evident but is not commonly severe. This phase may lead directly to an unrestricted suppurative encephalitis with the rapid development of severe headache, vomiting and drowsiness, as the result of increased intracranial pressure.

More commonly the suppurative process becomes localized as an abscess cavity with tough walls, through which there is little septic absorption. There is, therefore, no diffuse encephalitis, and the abscess produces signs and symptoms which depend upon its situation rather than its pathology.

In this, the common variety of cerebral abscess, the signs of increased intracranial pressure are, in the early stages, limited to occasional headache, by no means always severe, but often experienced on waking, and increased by stooping and coughing. Severe headache, vomiting, drowsiness, and slowing of the pulse, when they develop in such a case, probably mark the occurrence of mechanical obstruction to the outflow from the ventricles with internal hydrocephalus, and may be terminal in their onset. I would emphasize this point in view of the teaching in some quarters still current that headache and drowsiness, vomiting and slow pulse are the cardinal signs of cerebral abscess. In the variety of abscess that I am now discussing this is no more true than it is of cerebral tumour.

A recent case of cerebellar abscess will serve to illustrate this point. A man of 21, who had had chronic otitis media for many years, was admitted to hospital on account of occipital headache and profuse discharge from the left ear. He had been at work up till admission. Operation was performed the same day and pus evacuated from the mastoid. On the following day he made no complaint of headache, was alert and sensible, but was observed by the surgeon to have nystagmus on looking to the left and some inco-ordination of the left arm. The next day he complained suddenly of severe headache, began to vomit, and became drowsy. When I saw him twenty-four hours later his pulse had slowed from 75 to 55. He was incapable of speech and almost unconscious, being with difficulty roused to hold his arms out. When he did so the left fell rapidly away to the bed. There was bilateral ankle clonus and both plantar responses were extensor.

At the operation, two hours later, an ounce of pus was evacuated from the left lobe of the cerebellum. Within thirty-six hours the signs and symptoms of increased intracranial pressure had disappeared. The plantar responses were flexor, there was no complaint of headache, and he was as alert and bright as he had been on admission. The size of the abscess suggested that it had been present for two or three weeks.

In the early stages, therefore, we have to rely chiefly for diagnosis upon the signs of local damage caused by the abscess, together with complaint of intermittent headache. If the symptoms have supervened upon a mastoid operation and a full history is available, an account of fever, malaise and rapid pulse-rate persisting for a

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week or so after the operation, is also of value. Of the additional information to be obtained from lumbar puncture I shall speak later. The localizing signs of cerebral abscess may be considered under three headings: (1) cerebellar, (2) left temporal, (3) right temporal.

(1) Of the signs of *cerebellar* abscess in the early stages suboccipital headache is of some importance, but in the later stages, when internal hydrocephalus is developing, the headache is commonly bifrontal or general.

The abscess is usually situated deeply within the lateral lobe. Corresponding with this situation the symptoms are those of inco-ordination in the movements of the limbs on the same side, which is best shown in the upper limb. Disturbance of equilibrium and reeling gait are less common or later developments.

The simplest method of examination is by the finger—nose—finger test. On the affected side there is deviation from the line of movement, and a tendency to under- or over-shoot the mark, giving a general impression of clumsiness. Of the other tests, one of the best is falling away of the affected limb when support is withdrawn by the observer from beneath the outstretched hands of the patient.

The other sign of value is nystagmus. This is sometimes said to be absent by the otologist when its presence is recognized by the neurologist. The tendency of the eyes is to swing away from the side of the cerebellar lesion. This will show best when the patient is asked to look away from the direction of the spontaneous deviation, that is, towards the side of the lesion. If he is then asked to fix his gaze on the observer's finger, the eyes gradually—it may be quite slowly—swing back towards the mid-line. This spontaneous movement the patient may or may not correct. If he does so the quick component is added and nystagmus results. If there is no attempt at voluntary correction, one cannot say that nystagmus is present, but one may speak of an unwillingness in conjugate deviation towards one side, which as a unilateral sign is of almost as great localizing value as the complete nystagmus.

(2) In abscess of the *left temporal* lobe in a right-handed person the localizing sign of outstanding value is aphasia. If the abscess be situated far forward in the lobe it may not be present, but in the commoner site in the middle or posterior thirds it is an early sign, and easily elicited if the right method is employed. It is perhaps unnecessary at a meeting of this Section to emphasize the fact that a patient may be suffering from a definite aphasia and yet be able to converse without apparent difficulty and take part in the ordinary social life of a ward full of patients without the aphasia being discovered until the appropriate method of investigation is employed.

For instance [1], a boy aged 16, who had been under observation in hospital for a week, was suspected of having a left-sided cerebral abscess. The house surgeon, who was a good observer, had written a full report in which he stated that there was no aphasia. He had rightly concluded on other grounds that an abscess was present. We made a collection of a dozen heterogeneous objects and asked the patient to name them. Books, matches, pen, watch and half-a-dozen others were named correctly. There were only two failures. When shown a key he could describe its use but could not name it. He rejected all substitutes and promptly accepted the word "key" as correct; so also with a plate. He said it was to eat off, and made of china, but could not name it. As before, he rejected substitutes and accepted the correct name when it was given to him. The abscess was found in the middle of the left temporal lobe and he made a complete recovery.

The other signs of *temporal* lobe abscess are common to both sides. They are the only signs of a temporal abscess on the right side in a right-handed person. Of these there are three which are of practical value. The first is a slight weakness of the opposite side of the face, mostly of the lower half, which may be present when the patient talks or smiles, but is less apparent when he responds to the usual test of showing the teeth at command.

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The second group of signs are those which indicate pressure upon the pyramidal tract—absent or diminished abdominal reflexes, increased tendon-jerks or an extensor or doubtful plantar response.

The third sign I have placed last because it is less constantly present. When found it is of the greatest value—I refer to the defect in the visual field caused by involvement of the optic radiation. We owe to the anatomical researches of Adolf Meyer the knowledge that the optic radiation, as it leaves the thalamus, at first sweeps downwards and forwards and extends well forward into the temporal lobe before it turns back beneath and around the lateral ventricle to reach the occipital cortex. In this situation its strands are divided into two bundles, inferior and superior, which correspond with the upper and lower quadrants respectively of the visual field. The otitic abscess, as a rule, lies in the lower part of the temporal lobe, and is therefore likely first to affect the inferior bundle of the radiation, producing a homonymous defect in the superior quadrants of the opposite fields. This may be the only definite sign of a right temporal abscess, and the importance of examining the visual fields when such an abscess is suspected cannot be overrated. The examination can be made at the bedside in a few minutes with sufficient accuracy to discover a gross defect, even in a child who is seriously ill, as the following instance sufficiently illustrates:—

A girl aged 7 was admitted to hospital with a six weeks' history of intermittent headache, culminating in vomiting and drowsiness. There was a family history of phthisis, and a brother and a sister of the patient had died of tuberculous meningitis. There was a history of old-standing bilateral chronic otitis media. On examination the child was drowsy and occasionally put her hands to her head as if in pain.

Definite cervical rigidity was present; no skin or tendon reflexes were obtained.

Lumbar puncture gave a clear fluid which contained 32 cells per c.mm., all lymphocytes.

The provisional diagnosis was tuberculous meningitis. Rough examination of the visual fields, however, revealed a complete left homonymous hemianopia. Operation was performed a few hours later, and a large abscess discovered in the right temporal lobe, the pus containing streptococci. The child subsequently died of meningitis.

*The Cerebro-spinal Fluid in Cerebral Abscess.*

The fluid in a case of cerebral abscess is clear and contains a slight excess of cells, varying in my series of cases from 18 to 95 per c.mm. These are mainly lymphocytes. The protein content is increased—the chloride content is normal and sugar-reducing bodies are present. So long as the abscess remains localized this is the usual picture.

A preponderance of polymorphs in the fluid, with diminution of chlorides and absence of sugar reduction is usually associated with clinical evidence of meningitis and means that the abscess is leaking either into the ventricular system or into the subarachnoid space. The change in the cerebro-spinal fluid in such a case may be rapid.

For instance, in a patient with a right temporal abscess, who was mentally alert and whose general condition was fair, the fluid was clear and contained 32 lymphocytes per c.mm., .07 per cent. protein, and .71 per cent. chlorides. Next morning she suddenly became collapsed and unconscious. Lumbar puncture was done at once and gave an opalescent fluid full of polymorphs, which were not, however, counted. This patient was operated upon, but died three weeks later of meningitis. At autopsy both ventricles contained pus, and the cavity of the right ventricle was lined with granulation tissue. The moment of collapse probably marked the occurrence of leakage into the ventricle.

Estimation of the pressure in the cerebro-spinal fluid may also be of value in diagnosis, for a definite rise of pressure may sometimes be found preceding complaint of severe headache or other signs of increased intracranial pressure.

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## SUPERFICIAL ABSCESS.

Three years ago I showed before this Section, together with my colleague, Mr. W. H. Ogilvie, a case [2] in which, following otitis media and mastoiditis, a localized collection of pus formed upon the surface of the brain beneath the arachnoid membrane in the neighbourhood of the Sylvian fissure. This was successfully drained and the patient has since remained well. I have recently met with a similar case.

A child, aged 5, was admitted to hospital with one week's history of left otorrhœa; a mastoid operation was done the same day. At the operation the dura in relation to the roof of the antrum was injured. For ten days after the operation the child seemed well and was allowed up. On the eleventh day after operation, at 4 a.m., she had an attack of right-sided Jacksonian epilepsy. Thereafter, at short intervals, she had many more. I saw her at 11 a.m., when she was in a constant state of right-sided clonic convulsions, so that further examination was impossible. Lumbar puncture had been done at 8.30 a.m. The fluid was clear and contained a slight excess of lymphocytes.

The argument (1) from the epileptic attacks was that she had a cortical and, therefore, a superficial lesion; (2) from the spinal fluid that the infective focus was shut off from the general subarachnoid space. Mr. Gill Carey, who operated the same afternoon, made the following note:—

"There was a small patch of granulations present on the dura in relation to the roof of the antrum (the place where the dura was accidentally injured, but not penetrated, at the original operation). Watching these granulations carefully I saw a bead of pus come out, and on enlarging the opening with sinus forceps a quantity of pus shot out under considerable pressure. The amount of pus was enough to fill the cavity of the mastoid, roughly a drachm. There was no question of opening the dura, as it was already open. I should say, however, that the collection of pus was very superficial, as the cavity was a very shallow one. At the dressing after operation the forceps met with resistance after entering about  $\frac{1}{4}$  in."

After operation there was no recurrence of the fits. The child remained unconscious for two days—presumably an exhaustion effect of the epilepsy. There were no signs of generalized meningitis. When she was fit to be examined she had a right hemiparesis with aphasia and apraxia, which gradually cleared up. The wound healed in five weeks. Six months after this she appeared in good health, but was left with slight weakness of the right arm and leg.

I take it that in a case of this kind the pathway of the infection from ear to brain has for some reason become blocked at a point at which it has already become shut off from the general subarachnoid space, and would more usually progress into the cerebral substance. The infective process then spreads laterally in the plane in which it has become arrested. The process is slow, and in the neurological sense "silent," until it reaches above the Sylvian fissure to the face-arm area of the motor cortex.

Such cases are probably rare—their immediate recognition is of obvious importance. The danger is lest the convulsions be interpreted as the first sign of a generalized meningitis, and the chance of immediate operative relief be missed.

The distinguishing features are the sudden onset of convulsions or paralysis of cortical type and the findings in the cerebro-spinal fluid.

## LOCALIZED NON-SUPPURATIVE ENCEPHALITIS.

In the course of formation of every cerebral abscess there must be a pre-suppurative stage of inflammation in which there are engorgement, exudation and swelling without necrosis. I would suggest that, as in other tissues, the inflammatory process may become arrested at this stage and be resolved without pus formation.

That such a localized non-suppurative encephalitis may occur in relation to otitis media is perhaps doubtful. I put forward the hypothesis for discussion. It seems

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to me the most probable explanation of cases in which the signs of a cerebral abscess are at one time present but disappear without the evacuation of pus. I have had three such within my own experience.

The first was that of a boy who had a right-sided otorrhœa and mastoiditis, for which an operation was performed. I was asked to see him two or three weeks later on account of headache and drowsiness. I found slight swelling of both optic discs, which was confirmed by an ophthalmologist, and some alteration of the reflexes on the left side of the body, suggesting a right temporal lobe lesion. An exploratory operation was performed but no abscess found. As the symptoms persisted a second operation was done ten days later. No abscess was found, but the boy subsequently made a complete recovery.

The second case was that of a man with left-sided otitis admitted to hospital for headache and drowsiness. He was somewhat confused, was definitely aphasic and had a right homonymous hemianopia. He refused operation and was subsequently discharged from hospital, having completely lost his aphasia and hemianopia.

Of the third and most recent case I have more detailed notes.

A boy, aged 10, was admitted to hospital on September 11 on account of pain in and behind the right ear following an acute otitis media of one week's duration. The temperature next day was 100° F. and there was persistent pain with much discharge from the ear. At operation, the same day, pus was found in the mastoid cells and a small collection compressing the lateral sinus which was, however, not thrombosed. The dura in the middle fossa was exposed but not incised. The wound was drained. Following the operation there was a swinging temperature up in the evening between 99° and 105° F.; the pulse ranged from 90 to 120. Mentally he was noted by the sister in the ward to be odd, apathetic, not speaking unless spoken to, taking no interest in his toys. On the nineteenth day after the operation a doubtful extensor response was obtained from the left foot.

About this time he began to complain of right frontal headache, chiefly on waking in the morning. This was occasionally associated with nausea. I first saw him on October 7, twenty-five days after the operation, and found a definite extensor plantar response on the left with diminished abdominal reflexes on the same side. I made the diagnosis of right temporal abscess and operation was performed the same day. The dura was opened and the brain explored without any abscess being discovered. Lumbar puncture at the time of operation gave a clear fluid apparently under increased pressure, containing no increase of cells or protein.

Following this operation his condition remained much the same. The temperature and pulse-rate were still high, there was a good deal of complaint of headache, but, apart from this, apathy was the most noticeable feature. Six days after this second operation I found the physical signs unchanged, and in the light of my previous experiences I suggested that we might be dealing with a non-suppurative encephalitis. It was decided, therefore, that no further operation should be undertaken for the time being, but that he should be given full doses of hexamine.

About October 16, that is, five weeks from the date of admission, he began to improve, temperature and pulse fell and headache was less; he took a natural interest in his surroundings. On October 29 he seemed normal and I could no longer obtain an extensor response from the left foot. He has since remained well. It is, of course, possible that an abscess may yet be present, but I am inclined to the opinion that my provisional diagnosis of non-suppurative encephalitis will prove correct.

The point to which I want to draw attention in these three cases is that the encephalitis was in each sufficiently well localized to cause physical signs resembling those of a cerebral abscess. All were diagnosed as such, and the only one of the three who escaped operation was he who refused it. I have been interested to find, in a recent report [3] from the Mayo Clinic, by A. W. Adson, an account of three similar cases. All three were in children whose symptoms developed after otitis media. The first had signs of a right temporal abscess, with papilloedema, and progressive left facial weakness. The second also had papilloedema, with aphasia, right homonymous hemianopia, and right-sided weakness—the classical signs of a left temporal lesion. Both were explored, the first four, the second nine, weeks after the

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commencement of headache had first suggested the presence of intracranial trouble. In neither case was any abscess found. Both ultimately recovered. The third case, also, with signs suggesting a left temporal abscess, was watched for some weeks and the patient recovered without operation.

Adson offers no means of clinical distinction between what he calls the pseudo-brain abscess and the true. But the point of importance to be gained from these observations is that when the general and localizing signs of cerebral abscess are present a negative exploration does not necessarily mean an abscess missed. In such a case, therefore, it may be wise to await the possibility of spontaneous cure before proceeding to a secondary exploratory operation.

## REFERENCES.

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*Discussion.*—Dr. DAN MCKENZIE (President) said that Dr. Symonds had raised many points of novelty and interest. He (the speaker) had always particularly emphasized the points referred to in connexion with cerebellar abscess. It was usually stated that cerebellar abscess was more frequently missed than was temporo-sphenoidal abscess. If true, this seemed difficult to account for; he (Dr. McKenzie) wondered whether the point about nystagmus might not be one of the reasons. The books stated that in quite a large percentage of cases of cerebellar abscess no nystagmus was noticed. In some patients nystagmus was difficult to detect, but if the attention of a stuporose patient could be so directed that his eyes were turned towards the side of the lesion, a slow return to the neutral position would be noted, i.e., the slow component of the nystagmus was present, but not the quick one, because the stuporose condition prevented the operation of the higher nerve centres.

Dr. Symonds' reference to encephalitis was interesting, because cases were often seen in which there were symptoms apparently of brain abscess, which however was not found on exploration. Later on such patients nevertheless recovered. He (the speaker) had been called to see a boy who had a discharge from the ear, accompanied by headache and pain, and he had operated on the mastoid; he did not feel justified at that time in exploring the brain. Two days later, however, it was insisted that he should do so. The patient was semi-comatose, and the temporo-sphenoidal lobe was explored but with a negative result. The boy recovered.

There had recently been cases of encephalitis lethargica in which the symptoms had pointed to brain abscess. That diagnosis seemed to be confirmed if there was an ear discharge. With ordinary care there was no fear of harm from exploration of the brain.

In testing the integrity of the naming centre, Dr. Symonds had suggested giving many test objects. He (the speaker) had been accustomed to test with only two or three common articles, but apparently there was safety in a greater number. These points seemed trivial perhaps, but it was the summation of apparently trivial points that might make all the difference in a diagnosis.

Sir JAMES DUNDAS-GRANT said that Dr. Symonds' insistence on the signs of superficial abscess was a valuable point; he (Sir James) could look back on some puzzling cases and he now realized that they had been of that nature.

Mr. F. W. WATKYN-THOMAS said he had had a patient in whom for a short time, there had been pure motor aphasia, with a lesion of Broca's area on the left side. In this case nasal polypi had been removed, and the patient had come to the hospital having a fistula of the right frontal sinus and a superficial abscess. The right frontal sinus was opened and drained. The next day the patient had seemed well, but that night it was noticed that he "was talking nonsense." On the following morning he could say a few words, but they were unintelligible and disconnected. He could understand what was said to him, and could carry out suggested movements. There was right facial weakness, the tongue was protruded to the right, and

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there was a double Babinski's reflex. Lumbar puncture was performed, and two hours later a right brachial monoplegia developed and the patient was quite unable to speak. He could still turn in bed, make suggested movements, and so on. The arm area on the left side of the motor cortex was exposed, and a loculated subarachnoid collection of purulent cerebro-spinal fluid was found. Both frontal sinuses were widely opened. The left sinus was full of polypi, but there was no perforation of the inner wall. The patient died the following day. No track of the infection was found at the post-mortem examination, but there was a well-defined depression over the anterior portion of the left sylvian fissure, the foot of the rolandic area, and the third frontal convolution. He (Mr. Watkyn-Thomas) had seen several cases of injury of the area of Broca on the left side, but this was the only one in which aphasia had been present.

In none of the cases shown that day was there any evidence of crossed deafness. This fact was important, as it seemed to throw doubt on the existence of a contralateral hearing centre in the temporal lobe. In none of these cases, further, was there any sign of a "pure word deafness." In fact their evidence was against the existence of verbal images. Chatelin and de Martel had suggested the existence of a centre for stereognosis at the foot of the ascending parietal, but the evidence was, unfortunately, weak. If any reliance could be placed on it, such a sign would be valuable for localization.

Sir WILLIAM MILLIGAN said that according to statistics, temporo-sphenoidal abscess was twice as common as cerebellar abscess, but his own experience was just the opposite. One recalled earlier and less accurate days, when the diagnosis of "brain abscess" was made without any precise idea as to its location in the brain. Many surgeons operated on the temporo-sphenoidal lobe because statistics showed that to be the most likely site for abscess. A few years ago little was known about nystagmus, and for that reason cerebellar abscess was frequently missed.

With regard to the withdrawal and examination of cerebro-spinal fluid, in every case which he (the speaker) had seen in which there had been leakage into the ventricles, and an almost maniacal mental condition had developed, accompanied by a rise of temperature.

Did Dr. Symonds think that non-suppurative encephalitis was due to the effects of a circumscribed serous meningitis? Symptoms of pressure existed to such an extent as to lead to the idea that an abscess was present. He (Sir William) suggested that these symptoms were due to a localized condition and that there had been time for adhesions to form in the infected area, so that as a result of plastic inflammation the membranes became soldered together. Two years ago he (the speaker) had had a case in which he had the opportunity of watching developments. He operated on a young man who had developed a condition of pseudo-brain abscess. He became quite unconscious and passed both *fæces* and urine in bed. There were no symptoms enabling one to say definitely that he had a brain abscess, but as his condition was so grave, he (the speaker) explored the temporo-sphenoidal lobe. A good recovery ensued, and he had followed the case up with great interest ever since. It seemed to have been superficial damage, of inflammatory nature, of questionable origin, and he would like to hear what Dr. Symonds thought was the probable course of events.

Mr. G. J. JENKINS said that some years ago the late Mr. Hunter Tod had described a series of cases in which there was softening of brain, and in which at the operation he had been able to distinguish healthy brain substance from the infected area.

Dr. C. P. SYMONDS (in reply) said that sometimes cerebellar abscess caused him more difficulty in diagnosis than did temporo-sphenoidal, especially when labyrinthitis and cerebellar abscess co-existed. In the present week he saw a case in which the symptoms might be accounted for by labyrinthine irritation, and it was only discovered at the post-mortem examination that the patient had a cerebellar abscess as well. Another difficulty was that with the variety of cerebellar abscess to which Mr. Jenkins had referred, situated on the antero-inferior surface of the cerebellum, close to the jugular foramen, which might give very few localizing signs.

Mr. Watkyn-Thomas's case was interesting as a good example of superficial abscess, distant from the focus of infection but with a clue as to the track.

There was more risk from lumbar puncture in cerebellar abscess than in temporal abscess. If there was a serious increase of intracranial pressure with drowsiness and swelling of the optic discs, lumbar puncture was contra-indicated. The amount of fluid removed in a

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case should be small, 2 c.c. or 3 c.c. was sufficient. Because of the risk, he (Dr. Symonds) would not perform lumbar puncture in any case in which the diagnosis could be settled without it. That procedure should be reserved for cases in which there was doubt; in such cases it might afford just the information one was seeking.

In none of his (the speaker's) three cases of non-suppurative encephalitis had a leucocyte count been made, but in Adson's three cases a polymorphonuclear leucocytosis had been found such as would be expected in cerebral abscess, and therefore it was not a differentiating point.

Sir William Milligan had said that the symptoms in cases of so-called pseudo-brain abscess might be due to serous meningitis, but what he (the speaker) thought was that a collection of fluid on the surface sufficiently large to cause those symptoms must also be sufficiently large to be readily detectable at operation; and in the two cases of his own which were operated upon, and in Adson's cases, no such collection had been found. It took a large collection of fluid on the brain to cause aphasia, extensor plantar response on the opposite side and swelling of the optic discs. Further, in one case there had been involvement of the optic radiation, which could not have been caused by a superficial lesion. That was why he (Dr. Symonds) suggested the alternative explanation that these were cases of non-suppurative inflammation, something which never reached a breaking-down stage. He did not feel satisfied about this as a pathological proposition, because it did not take place in the ordinary way within the body. But it occurred in the skin, and an analogous instance could be found in amœbic hepatitis which often cleared up without any evacuation of pus.

Care should be taken in following up these cases. A patient might have a small brain abscess with a surrounding area of encephalitis, which might cause symptoms and signs, and as the abscess became more shut off the physical signs would diminish, though the abscess remained. Such an abscess might cause further symptoms, even after an interval of three years, as in a gunshot wound case of his own.

He had purposely not dealt with the atypical varieties of brain abscess, partly because he had not had much experience of them. He had seen one case of anterior temporal abscess with uncinat attacks, and two or three cases in which an abscess of the antero-inferior surface of the cerebellum was present without any of the inco-ordination of the limbs usually found in a deeply situated abscess of the lateral lobe.

## ILLUSTRATIVE CASES.

**Cerebellar Abscess.**

By R. J. CANN, L.R.C.P.Lond., M.R.C.S.Eng. (introduced by Mr. T. B. LAYTON).

R. A., MALE, aged 20.

In 1924, discharge from left ear. A polyp was removed—intermittent discharge afterwards.

Seen again November 4, 1926. Recurrence of aural discharge for one week, following a cold. During subsequent week complained of severe diffuse headache, frequent vomiting—became visibly thinner.

November 11, 1926.—Aural discharge profuse; drumhead obscured by granulation; no mastoid tenderness; slight facial weakness left side; no nystagmus; suspicion of paralysis of left external rectus muscle; reflexes normal; pointing error with left arm observed once, but not repeated; one observer thought nose-finger-nose test deficient on left side; headache now referred to occipital region; some rigidity of neck; temperature 100° F.

Same evening cortical mastoid operation by Mr. T. B. Layton; bone dense; pus in mastoid antrum; granulations on dura over transverse sinus; dura of middle and posterior fossæ exposed.